MINISTRY OF AGRICULTURE, FISHERIES AND FOOD

Polychlorinated biphenyl (PCB) residues in food and human tissues

The Thirteenth report of the Steering Group on Food Surveillance

REFERENCE ONLY
THIS BOOK MAY NOT BE
TAKEN OUT OF THE LIBRARY

Food Surveillance Paper No. 13

LONDON
HER MAJESTY'S STATIONERY OFFICE

SOUTHAMPTON UNIVERSITY LIBRARY

©Crown copyright 1983
First Published 1983

ISBN 0 11 242572 0

Contents

	Paras	*Pages*
Introduction	1–4	1
Production and Composition	5–7	2
Properties and Uses	8–9	3
Analytical Difficulties in Determining PCBs	10–11	4
Sources and Pathways in the Environment	12–16	5
Routes of Entry into Food	17–18	6
Levels of Intake of PCBs in the Average UK Diet . . .	19–21	6
Levels of PCBs in Fish and Shellfish	22–24	8
Levels of Intake of PCBs in Consumers of Large Quantities of Fish .	25	14
Consumers of Fish Liver Oils	26–27	15
Levels of PCBs in Animal Fats	28–29	15
Levels of PCBs in Milk	30–31	16
Miscellaneous Contamination of Food	32–34	17
Levels of PCBs in Human Fat	35–36	18
Levels of PCBs in Human Milk	37–38	18
Conclusions	39–41	19
References		20
Appendix I: Report of the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment		22
Appendix II: Consideration of the Report by the Food Additives and Contaminants Committee		27

Note: Summary of units used in this report
mg Milligram, one thousandth of a gram
µg Microgram, one millionth of a gram
mg/kg Milligrams per kilogram, equivalent to parts per million
µg/kg Micrograms per kilogram, equivalent to parts per thousand million

INTRODUCTION

1. Polychlorinated biphenyls (PCBs) are a group of chlorinated organic compounds whose properties and uses since the early 1930s have resulted in their broad dispersal into the environment. The realisation that they were environmentally stable and accumulated in the food chain led to action being taken in 1971 by the sole manufacturer of PCBs in the UK to restrict their use solely to closed systems such as transformers and capacitors. Similar action was taken in other countries following international agreement by OECD countries. In 1976 an EC directive was agreed (76/769/EEC) which placed the voluntary arrangements which already existed in the UK on a statutory basis throughout the EC. In 1977 the production of PCBs ceased altogether in the UK and the USA.

2. PCBs were first identified in environmental samples in Sweden in 1966 as interfering compounds which had been detected in the routine analysis of organochlorine pesticides. The possibility of adverse effects on human health from PCBs was highlighted in 1968 when 13 000 people in western Japan were found to have symptoms of poisoning ranging from skin disorders and hyperpigmentation to respiratory effects. These were attributed to eating rice-bran oil contaminated with commercial PCBs together with other impurities which had leaked from heat transfer equipment used in the manufacture of the oil. In 1969 the death of over 12 000 sea birds in the Irish Sea was attributed in part to high levels of PCBs found in their livers which were probably mobilised during a period of particular stress. In 1971 problems arose in the poultry industry in the USA when large numbers of chicken eggs failed to hatch. The cause was eventually traced to a toxic component of the fishmeal which formed part of the poultry feed. Further investigations revealed that leakage of PCBs, used as a heat-transfer fluid in the feed plant, had occurred into the fishmeal. As recently as 1979, in Idaho USA poultry and eggs were found to be contaminated with PCBs, through leakage of insulating fluid from a transformer. The source of contamination was traced to a meat packing plant in Montana which had sold contaminated feed, but not before the contamination had spread to 16 other States. A further outbreak of human poisoning by PCBs, again attributable to the consumption of contaminated rice oil, involving 2 000 individuals in Taiwan has recently been reported[1].

3. A similar though localised problem was identified in the UK during the autumn of 1979, when poultry from two packing stations in the West Country were found to be contaminated with PCBs. Thirty-five samples of broilers, one from a packing station in Devon and the rest collected from a packing station in Wiltshire, all had high levels of PCBs in abdominal fat, ranging from 0.2 to 4 mg/kg. It has not been possible to trace the source of the contamination but contaminated animal feed is thought to have been the most likely source. On 1 July 1980 Statutory Instrument 1980/638* came into operation specifically banning the use of PCBs as heat transfer fluids in plant used in food and feedstuff production. In addition it formalised the previously voluntary restriction on the use of PCBs in dispersive applications in papers, paints etc., which have given rise to a very significant proportion of the PCBs in the

---

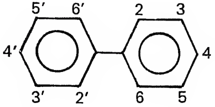
*'Control of Pollution (Supply and Use of Injurious Substances) Regulations'.

general environment in certain areas of the world. The implementation of this legislation should reduce the risk of contamination of food and animal feedstuffs with PCBs, although, since they may still be used in transformers and capacitors in food and feed industries, the risk of leakages or spillages occurring is not completely eliminated. The attention of the relevant industries has been drawn to this matter.

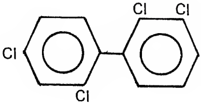
4. Checks on the PCB content of food in the UK have been made since 1966 when they were first found in wildlife. This report summarises the information that has been generated by a number of laboratories as part of their research and monitoring programmes and which have been collected together by the Panel for the Collection of Residue Data and the Working Party on Pesticide Residues for consideration by the Steering Group on Food Surveillance.

PRODUCTION AND COMPOSITION

5. PCBs are produced by the direct chlorination of biphenyl. There are 10 possible sites for chlorination on the biphenyl molecule and any particular polychlorinated biphenyl is named in accordance with the number of chlorine atoms in the molecule and their positions of substitution.



biphenyl



2,2′,3,4′-tetrachlorobiphenyl

There are 209 theoretically possible individual chlorinated biphenyls created by different substitutions and numbers of chlorine atoms (Table 1).

6. The biphenyl chlorination process produces mixtures of many PCBs. However, of the 209 possible PCBs, only about 100 are commonly found in commercial preparations. The industrial products are mixtures of these isomers and are sold on the basis of their average chlorine content. The only UK manufacturer of PCBs, the Monsanto Chemical Company, produced PCBs under the trade name 'Aroclor'. Aroclor products are referred to by a four-digit number e.g., Aroclor 1221 or Aroclor 1254. The first two digits refer to the 12 carbon atoms in the PCBs; the last two digits refer to the extent to which the particular Aroclor product is chlorinated (in weight per cent of chlorine). Thus Aroclor 1254 is identified as a complex mixture of chlorinated biphenyls, the entire mixture containing, by weight, 54 per cent chlorine. An exception to this is Aroclor 1016 which in fact contains 41 per cent chlorine. Pentachlorobiphenyls provide the major proportion of the higher chlorinated material normally found nowadays in commercial products.

Table 1
Description of the 209 possible chlorinated biphenyl isomers

Compound	Number of chlorine atoms in molecule	Number of possible isomers in this category	Per cent chlorination by weight*
Biphenyl	0	(1)	0
Monochloro biphenyl	1	3	18.79
Dichloro biphenyl	2	12	31.77
Trichloro biphenyl	3	24	41.30
Tetrachloro biphenyl	4	42	48.56
Pentachloro biphenyl	5	46	54.30
Hexachloro biphenyl	6	42	58.93
Heptachloro biphenyl	7	24	62.77
Octachloro biphenyl	8	12	65.98
Nonachloro biphenyl	9	3	68.73
Decachloro biphenyl	10	1	71.18
		209	

*Based on Cl = 35.45

7. The PCB homologues demonstrate a gradation of properties and their stability increases as the chlorine content increases. Water solubility of PCBs decreases with increasing chlorine content and studies on PCBs with 1, 2, 4 or 5 chlorine atoms have shown that they are more readily metabolised in mammals and birds than those with six or more chlorine atoms[2].

PROPERTIES AND USES

8. The physico-chemical properties of PCBs which account for their previous widespread industrial use are summarised in Table 2. At room temperature commercial mixtures of PCBs are mobile oils (Aroclors 1221, 1232, 1242, 1248), viscous liquids (Aroclor 1254) or sticky resins (Aroclors 1260 and 1262); viscosity increasing with increasing chlorine content of the mixture. Some examples of commercial applications of PCBs prior to the 1976 EC directive are listed in Table 3. Most of these uses are now restricted throughout the EC.

Table 2
Main physico-chemical properties of PCBs

High boiling points
Low water solubility
High dielectric constant
Excellent thermal stability
High resistance to acidic and basic hydrolysis
Fire resistant
Good thermal conductors
Good lubricating characteristics

Table 3
Some industrial applications of PCBs

Dielectric in capacitors and transformers
Immersion oils
Cutting oils
Heat exchange fluids
Hydraulic fluids
Paints
Plasticizers
Printing inks
Fire retardants
Carbonless copy paper

9. In a survey carried out amongst OECD countries in 1971, it was found that the major use of PCBs was as dielectrics in capacitors and transformers[3]. In 1972 sales were voluntarily restricted in the UK and USA to the use of lower chlorinated PCBs only as dielectrics in enclosed systems and limitations on sales were subsequently introduced in other countries.

ANALYTICAL DIFFICULTIES IN DETERMINING PCBs

10. The complexity of extraction, clean up, isolation and quantification of PCBs had presented an acute analytical problem and was a reason for their delayed detection in the environment. The major difficulties in the determination of PCBs are to separate them from other organochlorine compounds including pesticides, and to derive a single quantitative figure from a variable mixture of many components. A schematic outline of a commonly used method for the analysis of PCBs is shown in Fig 1.

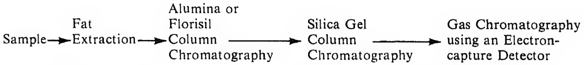

Sample
Fat Extraction
Alumina or Florisil Column Chromatography
Silica Gel Column Chromatography
Gas Chromatography using an Electron-capture Detector

Fig 1. Schematic outline of analytical method for PCBs.

11. The sensitivity of the analytical method used is affected by the degree of chlorination and is not equal for all the PCB components[4]. This does not lead to major difficulties when the sample under investigation has been directly contaminated by a commercial PCB mixture, as that mixture can be used to standardize the response of the apparatus i.e. allow for the comparison of peak heights and subsequent quantification. However, difficulties are encountered when PCBs have undergone selective environmental degradation or selective metabolism by animal tissues. Studies of the tissue distribution of PCBs after administration of commercial mixtures have observed a reduction in the amounts of the lower chlorinated biphenyls detected[2]. In such situations an attempt at quantification is only possible by comparison with the commercial mixture which produces a pattern of peaks most nearly resembling the peaks produced from the sample. Different laboratories choose different standards

according to the pattern of peaks most usually encountered in their samples. With the exception of those samples where major differences are observed standard commercial formulations are used to quantify PCB residues. The data reported by the Laboratory of the Government Chemist in this report were almost all quantified by comparison with Aroclor 1260 as the standard whereas the fish data are usually quantified by comparison with Aroclor 1254.

SOURCES AND PATHWAYS IN THE ENVIRONMENT

12. The estimated cumulative world production of PCBs since 1930 is between one and two million tonnes[2,5]. Of this more than one half has entered dumps and landfills, where it is likely to be stable and released only very slowly. PCBs have also entered the environment by the disposal of industrial fluids into rivers and coastal waters, by leakage from non-enclosed systems, or by volatilization into the atmosphere from the incineration of PCB-containing material.

13. The voluntary control on the use of PCBs in open systems introduced progressively in the 1970s, has limited the rate of escape into the environment but PCBs are expected to continue to be detected in the environment mainly due to:

(a) accidental leaks and spillages from the servicing of industrial equipment (transformers etc);

(b) incomplete destruction during the combustion of products containing PCBs (very high combustion temperatures (1200°C) are required for complete and rapid decomposition);

(c) escape from plants where PCBs are reclaimed from redundant equipment or processed into other products;

(d) leachate from landfill disposal sites and industrial discharge;

(e) the breakdown of scrap electrical condensers where PCBs have not been recovered;

(f) recycling of PCB-containing papers in the production of paper from scrap paper;

(g) disposal of sewage sludge.

14. In accordance with EC directive (76/403/EEC) the uncontrolled discharge, dumping and tipping of PCBs is prohibited. Member States have to set up or designate installations or establishments authorised to dispose of PCBs. There are five high-temperature incinerators licensed under the Control of Pollution Act 1974 in the UK. They are also registered under the Alkali Acts for control of atmospheric emissions, and most of the currently generated PCB wastes are disposed of at these units. The disposal of small capacitors to landfills or scrap recovery facilities is not specifically restricted by the directive and the Department of the Environment's Waste Management Paper No 6 recommends that such disposal practice may continue provided the concentration is not more than one capacitor per tonne of landfilled domestic refuse (approximately equivalent to 300–400 grams per 1000m^3 of refuse). It is estimated that approximately 600 tonnes of PCBs derive from disposal of small capacitors per annum, but the development of alternative capacitors will lead to a decrease in PCB-waste production to 100 tonnes per annum by 1990[6].

15. In the past wastes containing PCBs have been disposed of to landfills. Between 1965 and 1970 waste from PCB manufacture in the UK was disposed into two quarries at Maendy and Brofiskin in Wales. It was suspected that PCBs could contaminate the leachate from the quarries and samples of ground water and surface water have been sampled regularly at both sites since 1973. At Maendy the maximum concentration of PCBs in surface leachate in 1973 was 0.5 μg/l. At Brofiskin between 1973 and 1976 the concentration of PCBs in surface leachate ranged from 0.02 to 0.1 μg/l. Samples of water were also taken on a regular basis from springs within 1.5 km of the site (a total of five at Maendy, and six at Brofiskin) to determine whether pollution of groundwater had occurred within the aquifers beneath the landfill sites. No pollution attributable to the landfills was found in the spring water of either site[7,8].

16. The possibility that animals on farms in the vicinity of the quarries might have accumulated residues of PCBs in their tissues as a result of access to contaminated water or feed was also examined. Milk was sampled from farms in each of the areas of the two quarries and from a control farm in an entirely different area. Some samples of animal tissues were also taken, including some from a sheep which had been raised on pasture adjacent to the quarry. In every case the residues in tissues were at low levels.

ROUTES OF ENTRY INTO FOOD

17. Foods have become contaminated with PCBs by three main routes:

(a) Direct contamination of food or animal feedstuffs by an industrial accident;

(b) Migration from packaging into food (see paragraph 32); and

(c) Absorption from the environment by fish, birds and other animals.

18. Attempts have been made to restrict the contamination from routes (a) and (b) and there are now no food or feed processing plants in the UK which use PCBs as heat transfer fluids. However, the third route is difficult to control. As PCB production has only been restricted since 1977, it has been estimated that levels in the environment are unlikely to decrease in the next 20 years and could increase in the short term. Route (c) will therefore continue to be the main source of PCBs in the UK diet.

LEVELS OF INTAKE OF PCBs IN THE AVERAGE UK DIET

19. An estimation of the dietary intake of PCBs by the general population in the UK has been made by means of total diet studies. Samples were analysed for PCBs in 1970–71, 1974 and 1976 (Table 4).

20. Since the majority of total diet samples analysed did not contain detectable levels of PCBs, it has not been possible to quantify the intake accurately. However, it is estimated that the *per capita* daily intake of PCBs from food in the UK is not likely to be greater than 40 micrograms and is probably below 10 micrograms.

21. Not surprisingly, in view of the known affinity of PCBs for lipid-rich foods, the PCBs were found with greatest frequency in milk and meat and particularly in the fish group. Programmes of more detailed sampling of the foods in these groups have therefore been undertaken.

Table 4

PCBs in total diet samples (expressed as mg PCBs per kg)

Only the data for those towns where certain food groups were found to contain quantifiable PCB residues are shown below:

1. Fourth quarter — 1970

Town	Cereals	Meat and Fish	Fish	Fats	Fruits and Preserves	Root Vegetables	Other Vegetables	Milk	Weighted mean for a whole diet
Aberdeen	*	*	*	*	*	*	*	0.002	< 0.01
Glasgow	*	*	*	*	*	*	*	0.002	< 0.01
Newcastle	0.01	*	*	*	*	*	*	†	< 0.01
Norwich	0.1	*	*	*	*	*	*	†	< 0.04
Sheffield	*	*	*	*	*	*	*	0.002	< 0.01

2. First quarter — 1971

Aberdeen	*	*	*	*	*	*	*	0.002	< 0.01
Brighton	*	*	*	*	*	*	*	0.002	< 0.01
Liverpool	*	*	*	*	*	*	*	0.002	< 0.01
London	*	0.02	*	*	*	*	*	†	< 0.01
Norwich	0.05	*	*	*	*	*	*	†	< 0.02
Torquay	*	*	*	*	*	*	0.01	†	< 0.01

3. Second quarter — 1971

Belfast	*	*	0.02	*	*	*	*	†	< 0.01
Cardiff	*	*	0.01	*	*	*	*	†	< 0.01
Cookstown	*	*	0.06	*	*	*	*	†	< 0.01
Jersey	*	*	0.01	*	*	*	*	†	< 0.01
Newcastle	*	*	0.03	*	*	*	*	†	< 0.01
Norwich	0.04	*	0.02	*	*	*	*	†	< 0.02
Torquay	*	*	0.05	*	*	*	*	†	< 0.01

4. First quarter — 1974

Bristol	*	0.02	0.01	*	*	*	*	†	< 0.01
Cardiff	*	*	*	*	*	*	*	0.003	< 0.01
Edinburgh	*	0.01	0.02	0.04	*	*	*	†	< 0.01
Glasgow	*	0.01	*	*	*	*	*	†	< 0.01
Newcastle	*	*	0.02	0.04	*	*	*	0.005	< 0.02
Shrewsbury	*	*	0.04	*	*	*	*	†	< 0.01

5. Second quarter — 1974

Town	Cereals	Meat and Fish	Fish	Fats	Fruits and Preserves	Root Vegetables	Other Vegetables	Milk	Weighted mean for a whole diet
Brighton	*	0.02	0.02	0.03	*	*	*	†	< 0.02
Bristol	*	0.01	0.02	0.05	*	*	*	0.003	< 0.02
Ilkley	*	0.01	*	*	*	*	*	†	< 0.01
Liverpool	*	0.02	0.01	*	*	*	*	†	< 0.01
Sheffield	*	0.02	0.01	*	*	*	*	†	< 0.01
Torquay	*	*	0.09	*	*	*	*	†	< 0.01
Worcester	*	*	0.09	*	*	*	*	†	< 0.01

6. Fourth quarter — 1974

Aberdeen	*	0.01	*	*	*	*	*	†	< 0.01
Belfast	*	*	0.02	*	*	*	*	†	< 0.01
Brighton	*	0.01	0.02	*	*	*	*	0.003	< 0.01
Cardiff	*	*	0.04	*	*	*	*	†	< 0.01
Edinburgh	*	*	0.01	*	*	*	*	†	< 0.01
Ilkley	*	0.04	0.03	*	*	*	*	0.006	< 0.02
Liverpool	*	*	0.01	*	*	*	*	†	< 0.01

7. First quarter — 1976

Cardiff	*	*	0.01	*	*	*	*	†	< 0.01
Jersey	*	*	0.01	*	*	*	*	†	< 0.01
Shrewsbury	0.05	*	0.02	*	*	*	*	†	< 0.02

8. Second quarter — 1976

Cookstown	*	*	0.02	*	*	*	*	†	< 0.01
Leicester	*	0.01	0.01	*	*	*	*	†	< 0.01
Sheffield	*	*	0.01	*	*	*	*	†	< 0.01

9. Third quarter — 1976

Brighton	*	0.01	0.01	*	*	*	*	†	< 0.01
Shrewsbury	*	*	0.01	*	*	*	*	†	< 0.01

**PCB concentration less than reporting limit 0.01 mg/kg*
†PCB concentration less than reporting limit 0.002 mg/kg

LEVELS OF PCBs IN FISH AND SHELLFISH

22. Research has shown that the concentration of PCBs can be increased by up to 100 fold through each predator in the marine food chain system. Because of the length of this system levels of up to 10^7 times higher than those measured initially in the water are ultimately achieved[9]. Direct uptake from the water is also a significant route of entry and this can account for a substantial part of the total body burden. Fish are therefore particularly susceptible to contamination by PCBs and have been monitored as a potentially important source of PCBs in the diet.

23. Levels of PCBs in fish and shellfish landed from UK marine waters have been monitored since 1970. Data obtained from these surveys are summarised in Tables 5 and 6. Fish muscle and liver were analysed separately and generally PCB concentrations increased with increasing tissue lipid content. Highest concentrations of PCB residues were found in whiting liver.

Table 5

Levels of PCBs in marine fish caught around UK coasts 1970–1979[10–15]

Species	Tissue	Year	Residues of PCBs (mg/kg wet weight)		No. of analyses*
			Range	Mean	
Cod	Muscle	1970	0.01 to 0.05	NA	NA
		1971	0.01 to 0.05	NA	NA
		1972	< 0.02 to 0.07	NA	NA
		1973	< 0.02 to 0.07	NA	NA
		1974	< 0.01 to 0.04	0.02	10
		1975	< 0.05 to 0.07	0.05	13
		1976	< 0.05 to 0.05	0.05	13
		1977	< 0.05 to 0.92	0.18	10
		1978	< 0.05	< 0.05	2
		1979	< 0.05	< 0.05	2
	Liver	1970	0.4 to 12	NA	NA
		1971	0.5 to 12	NA	NA
		1972	1.1 to 18	NA	NA
		1973	2.0 to 18	NA	NA
		1974	1.4 to 13	5.1	10
		1975	3.1 to 15	5.4	13
		1976	0.5 to 26	7.0	16
		1977	0.09 to 2.4	1.6	10
		1978	1.7	1.7	1
		1979	0.27 to 2.9	1.4	6
Plaice	Muscle	1970	0.01 to 0.14	NA	NA
		1971	< 0.01 to 0.17	NA	NA
		1972	< 0.02 to 0.19	NA	NA
		1973	< 0.01 to 0.09	NA	NA
		1974	0.02 to 0.3	0.08	5
		1975	< 0.05 to 0.31	0.09	19
		1976	< 0.05 to 0.3	0.09	16
		1977	< 0.05 to 0.1	0.06	11
		1978	< 0.05 to 0.17	0.10	5
	Liver	1970	0.02 to 1.6	NA	NA
		1971	< 0.02 to 3.2	NA	NA
		1972	< 0.05 to 9.7	NA	NA
		1973	0.02 to 1.9	NA	NA
		1974	0.08 to 2.0	0.7	5
		1975	0.09 to 5.5	1.2	19
		1976	< 0.05 to 4.2	0.82	19
		1977	< 0.05 to 0.82	0.31	10
		1978	0.04 to 1.5	0.55	14
		1979	0.05 to 1.3	0.71	7

Table 5 cont.

Species	Tissue	Year	Residues of PCBs (mg/kg wet weight)		No. of analyses*
			Range	Mean	
Whiting	Muscle	1970	< 0.01 to 0.14	NA	NA
		1971	< 0.01 to 0.74	NA	NA
		1972	0.02 to 0.42	NA	NA
		1973	< 0.02 to 0.04	NA	NA
		1974	0.05 to 0.07	0.06	2
		1975	< 0.05 to 0.07	0.05	13
		1976	< 0.05 to 0.1	0.06	13
		1977	< 0.05 to 0.07	0.06	2
		1979	< 0.05	< 0.05	2
	Liver	1970	0.35 to 14	NA	NA
		1971	0.64 to 28	NA	NA
		1972	1.1 to 26	NA	NA
		1973	2.9 to 15	NA	NA
		1974	2.5 to 7.6	5.1	2
		1975	2.6 to 16	6.5	14
		1976	0.2 to 20	5.5	17
		1977	0.7 to 5.7	2.8	3
		1978	2.3	2.3	1
		1979	11 to 15	13	2
Herring	Muscle	1970	0.06 to 0.34	NA	NA
		1971	0.10 to 0.63	NA	NA
		1972	0.09 to 0.53	NA	NA
		1973	0.07 to 1.1	NA	NA
		1974	0.18	0.18	1
		1975	0.08 to 0.9	0.22	7
		1976	0.05 to 1.2	0.28	6
	Liver	1970	< 0.01 to 0.54	NA	NA
		1971	0.10 to 0.48	NA	NA
		1972	0.1 to 0.54	NA	NA
		1973	0.1 to 0.44	NA	NA
		1974	0.15	0.15	1
		1975	0.08 to 1.5	0.58	3
		1976	0.01 to 1.9	0.38	6
Mackerel	Muscle	1970	0.11 to 0.5	NA	NA
		1971	< 0.10 to 0.68	NA	NA
		1972	0.06 to 1.7	NA	NA
		1973	0.1 to 0.2	NA	NA
		1974	0.55	0.55	1
		1975	0.18 to 0.6	0.36	5
		1976	0.07 to 0.7	0.28	7
		1977	0.14 to 1	0.57	2
		1978	0.57	0.57	1

Table 5 cont.

Species	Tissue	Year	Residues of PCBs (mg/kg wet weight)		No. of analyses*
			Range	Mean	
Mackerel	Liver	1970	0.25 to 1.8	NA	NA
		1971	0.07 to 1.9	NA	NA
		1972	0.2 to 3.5	NA	NA
		1973	0.17 to 0.76	NA	NA
		1974	0.50	0.50	1
		1975	0.46 to 1.6	1.1	5
		1976	0.06 to 2.5	0.74	7
		1977	0.57 to 8	4.3	2
		1978	0.46	0.46	1
Sole	Muscle	1974	0.01 to 0.19	0.1	2
		1975	< 0.05 to 0.25	0.13	4
		1976	< 0.05	< 0.05	13
		1977	< 0.05	< 0.05	4
	Liver	1974	0.42	0.42	1
		1975	0.25 to 1.4	0.67	4
		1976	0.09 to 1.0	0.36	17
		1977	0.4 to 1	0.63	4
Dab	Muscle	1974	< 0.02 to 0.66	0.34	2
		1975	< 0.05 to 0.3	0.11	6
		1976	< 0.05 to 0.13	0.08	5
	Liver	1975	0.2 to 4.6	1.5	6
		1976	0.24 to 1.9	1.1	11
Bass	Muscle	1974	0.05	0.05	1
	Liver	1974	1.3	1.3	1
Dogfish	Muscle	1974	0.05 to 0.18	0.16	4
		1975	0.08 to 0.14	0.11	3
		1976	< 0.05 to 0.05	0.05	3
		1979	0.09	0.09	1
	Liver	1974	0.43 to 18	6.2	4
		1975	2.2 to 8.8	6.5	3
		1976	3.2 to 11.4	6.8	4
		1977	6.2 to 7.6	6.9	2
		1979	6.8	6.8	1
Flounder	Muscle	1976	0.05 to 0.15	0.08	3
		1977	< 0.05 to 1	0.26	6
	Liver	1976	0.09 to 0.56	0.37	3
		1977	0.14 to 4	1.3	7
Pout Whiting	Muscle	1974	0.01 to 0.03	0.02	3

SOUTHAMPTON UNIVERSITY LIBRARY

Table 5 cont.

Species	Tissue	Year	Residues of PCBs (mg/kg wet weight)		No. of analyses*
			Range	Mean	
Ray	Muscle	1974	0.02	0.02	1
		1975	< 0.05 to 0.07	0.05	6
		1976	< 0.05	< 0.05	5
		1977	< 0.05	< 0.05	2
	Liver	1974	2.4	2.4	1
		1975	1.5 to 3.9	2.5	6
		1976	0.58 to 6.8	2.5	7
		1977	1 to 3	2	2
Sea bream	Muscle	1974	0.03	0.03	1
	Liver	1974	0.16	0.16	1
Sprats	Muscle	1974	0.03	0.03	1
Haddock	Muscle	1974	< 0.01	< 0.01	3
	Liver	1974	1.3 to 1.6	1.5	3
Witch	Muscle	1974	0.08	0.08	1
	Liver	1974	0.17	0.17	1
Yellow gurnard	Adipose	1971	2.2 to 5.7	3.7	7
	Muscle	1971	0.03 to 0.12	0.05	10
	Liver	1971	0.18 to 1.7	1.3	10

NA not available.

*The number of fish within each sample that was analysed varied between 1 and 20.

24. PCBs have been found to be ubiquitous contaminants of freshwater fish in English and Scottish rivers and lochs (Table 7). PCB levels were generally highest in species and tissues with a high lipid content; maximum levels were found in pike liver. However, freshwater fish livers are not normally consumed. The highest residues found in the samples of freshwater fish muscle analysed did not exceed 1mg/kg. Very much higher PCB residues levels have been reported in freshwater fish in those countries where there are large land-locked bodies of freshwater receiving industrial or sewage pollution; for example carp in Lake Michigan have been found to contain 52mg/kg PCBs and fish from the Great Miami River, which receives discharges from paper mills, contained more than 150 mg/kg PCBs[16].

Table 6

Levels of PCBs in UK shellfish species 1970–1976[10–13]

Species	Tissue	Year	PCB residues (mg/kg wet weight)		No. of analyses
			Range	Mean	
Whelks		1970–74	< 0.01 to 0.24	0.08	19
Pink shrimps		1970–74	0.02 to 0.24	0.06	10
Brown shrimps		1970–74	< 0.01 to 0.4	0.06	8
		1975	0.07 to 0.2	0.12	7
Queens	Muscle	1970–74	0.01 to 0.09	0.05	2
	Gonad		< 0.01	< 0.01	1
	Digestive gland	1971	0.07 to 0.24	0.15	4
	Adductor muscle		0.02 to 0.05	0.03	4
Oysters		1970–74	0.05 to 0.36	0.10	NA
Mussels		1970–74	0.01 to 0.44	0.14	NA
		1975	< 0.01 to 0.28	0.12	7
		1976	< 0.01 to 0.1	0.03	11
Crabs	Body	1970–74	< 0.01 to 6.0	0.75	NA
	Claw		< 0.01 to 2.8	0.13	NA
Lobsters	Body	1970–74	1.6	1.6	1
	Claw		< 0.01	< 0.01	1
	Tail		0.01 to 0.08	0.04	2
Cockles		1970–74	< 0.01 to 0.02	< 0.01	8
Crawfish	Body	1970–74	0.24	0.24	1
	Tail		0.06	0.06	1
Squid	Mantle musculature	1971	0.08 to 0.18	0.13	5

NA = not available.

Table 7

Levels of PCBs in freshwater fish in UK

Species	Origin (no. of sites from which samples taken)	Year	Tissue	No. of samples	Range of PCBs (mg/kg wet weight)
Brown trout	Scotland (30)	1972	muscle	150	0.01 to 0.22
			liver	150	0.02 to 2.5
	England (1)	1976	muscle	2	0.03 to 0.1
			liver	2	0.11 to 0.13
Sea trout	Scotland (10)	1972	muscle	60	0.03 to 0.41
Perch	Scotland (2)	1972–75	muscle	50	0.01 to 0.09
Eels	Scotland (2)	1973	muscle	9	0.29 to 0.69
		1977	muscle	12	0.18 to 0.8
Pike	England (6)	1976	muscle	27	0.01 to 0.34
			liver	26	0.03 to 11.7
Bream	England (1)	1976	muscle	4	0.13 to 1.0
			liver	4	0.21 to 0.94
Roach	England (1)	1976	muscle	4	0.02 to 0.08
			liver	3	0.19 to 0.33

LEVELS OF INTAKE OF PCBs BY CONSUMERS OF LARGE QUANTITIES OF FISH

25. The only food group in total diet studies (paras 19 to 21) consistently found since 1971 to contain PCB residues is the fish group. The possibility exists therefore that fish consumers whose intake is well above the national average could be exposed to high levels of PCBs in their diet and this possibility was investigated by dietary studies carried out in 1977. Forty-eight diets from people living in fishing communities bordering the NE Irish Sea and the English Channel, in which individual fish consumption exceeded 0.5 kg per week, were examined. Concentrations of PCBs ranged from 0.01 to 0.35 mg/kg with a mean of 0.1 mg/kg. The total weekly intake was found to range from 6–260 μg (0.09-3.7 μg/kg body weight) with a mean of 74 μg (1 μg/kg body weight) from fish consumption.

CONSUMERS OF FISH-LIVER OILS

26. Tables 5 to 7 show that PCBs are found in highest concentrations in lipid-rich fish tissues i.e. fish livers. Consumption of fish liver oils could lead to high intakes of PCBs and retail samples of these products were therefore analysed for PCBs. Four samples of bottled cod liver oil, two of halibut liver oil capsules and one of bottled halibut liver oil were analysed:—

Cod liver oil (170 ml bottles): 0.9, 1.0, 1.1, 1.2 mg/kg
Halibut liver oil (capsules): 0.7, 0.7 mg/kg
Halibut liver oil (5 ml bottle): 7 mg/kg

The data for halibut liver oil demonstrates the variation that can occur. Similar findings were reported in 1972 when two samples of halibut liver oil capsules were examined; it was estimated that one contained 1.8 mg PCB per kg, while the other contained 6 mg/kg.

27. From these data, if an infant were to receive daily vitamin supplementation by means of cod or halibut liver oil (approximately one teaspoon), rather than by drops containing synthetic vitamins A and D, its PCB intake from this source would be between 1 and 7 μg/day.

LEVELS OF PCBs IN ANIMAL FATS

28. Home-produced and imported meat samples have been monitored for PCBs since 1968. The results are summarised in Table 8.

29. These surveys indicate that PCBs are generally undetected or at low levels; a smaller proportion of the home-produced meat than the imported meat contained detectable residues.

Table 8

Levels of PCB in the kidney fat of home-produced and imported carcases 1968–1977

1. **Lamb**

Origin	No. of samples analysed	Year	Residues of PCBs (mg/kg)	
			Range	No. of positives
Home-produced	53	1969	< 0.01 to 0.3	1
	35	1970	< 0.01 to 0.07	1
	23	1974	< 0.01 to 0.06	5
	89	1975	< 0.01 to 0.15	21
	64	1976	0.01 to 0.07	14
New Zealand	7	1977	< 0.01	0

Table 8 cont.

2. **Beef**

Origin	No. of samples analysed	Year	Residues of PCBs (mg/kg)	
			Range	No. of positives
Home-produced	42	1968–69	< 0.01 to 0.07	2
	22	1975	< 0.01 to 0.14	11
	7	1976	< 0.01 to 0.05	3
Holland/Denmark	8	1975	< 0.01 to 0.2	6
Germany/America/ Botswana	18	1976	< 0.01 to 0.1	9
Germany/Botswana/ Denmark/New Zealand	38	1977	< 0.01 to 0.1	11

3. **Veal**

Origin	No. of samples analysed	Year	Residues of PCBs (mg/kg)	
			Range	No. of positives
Holland	22	1974	0.01 to 0.19	18
Holland	18	1975	trace to 0.12	18
Holland	19	1976	< 0.01 to 0.06	11
Holland	16	1977	< 0.01 to 0.07	6

LEVELS OF PCBs IN MILK

30. Peaks suggestive of the presence of PCBs were recognised on the chromatograms given by bulk milk samples in early 1967. These peaks were relatively insignificant, but increased in prominence as the year progressed when four of 37 samples were reported as containing PCBs (see Table 9). The bulk samples continued to show occasional PCB-type traces during 1968, but the levels did not exceed an estimated 0.002 mg/kg on a whole milk basis. Again, during 1969, PCB-type traces occurred erratically, with an estimated maximum level of 0.003 mg/kg. Routine PCB analysis of bulk milk samples ceased in 1969 but milk samples collected as part of total diet studies were analysed for PCBs from 1970 onwards.

31. Only a small number of positive samples have been found with low residue levels which give no cause for concern. There has been no discernible trend in levels of PCBs in milk over the 12 years of monitoring.

Table 9

PCB residues in milk

Year	No. of samples	Range (mg/kg)	No. of positives	Mean of positives (mg/kg)
1967	37	< 0.002 to 0.015	4	0.006
1968	76	< 0.002 to 0.002	NA	NA
1969	60	< 0.002 to 0.003	NA	NA
1970	8	< 0.002 to 0.002	3	< 0.002
1971	34	< 0.002 to 0.002	3	< 0.002
1974	23	< 0.002 to 0.006	5	0.004
1976	8	< 0.002	0	—
1978	7*	< 0.001 to 0.002	1	0.002

NA not available

* Samples of milk taken from dairy farms whose pastures had received sewage sludge.

MISCELLANEOUS CONTAMINATION OF FOOD

32. There have been incidents in other countries when food packaging materials have become contaminated with PCBs as a result of their having been made from recycled paper which has included 'carbonless' copy paper containing PCBs. Thirteen specimens representing all the types of 'carbonless' copy paper available were examined by the Laboratory of the Government Chemist in 1970. No evidence of PCBs was found in nine of the specimens. Two contained quantities of the order of a few per cent but these papers are now no longer in production[17]. The replacements for the samples containing positive levels were also examined and only minute traces were detected which may indicate the carry-over of PCBs from contaminated vessels or machinery. It seems very unlikely that further contamination of food will result from this source and the European paper and packaging industries have applied controls to ensure that PCBs are no longer present in their products.

33. Contamination of materials which come in contact with food was investigated by the Laboratory of the Government Chemist in 1972. Twenty-five samples, as listed in Table 10, were examined for the presence of PCBs. Much background interference was present on the glc traces, so that the lower limit of detection was about 1 mg PCBs per kg. No PCBs were detected in any sample above this level. In addition a specimen of wax used for sealing jars of Stilton

Table 10

Number of samples of materials which come into contact with food which were analysed for PCBs

Wrappers in direct contact with food: paper	14
Wrappers in direct contact with food: cellophane	2
Outer wrappers for food: cartons	3
Milk carton	1
Brown paper	1
Cardboard	1
Newspaper	1
Photocopying papers	2

cheese was found to contain about 1 mg PCB per kg. In use the wax seal was separated from the cheese by a paper disc and no PCBs were detected in that portion of the cheese which was in direct contact with the paper.

34. Contamination of the human food chain may arise from the recycling of sewage sludge containing PCBs onto agricultural land, either by direct contamination of the crops or by uptake of PCBs from the soil. In the USA between 1971 and 1975, 69 samples of sludges were examined for PCBs; concentrations ranged from below the limits of detection up to 352 mg/kg[18]. A Canadian study[19] had demonstrated the uptake of PCBs by some sludge-treated crops and as a result it was decided to investigate the position in the UK. Table 9 includes limited data from a study of milk from seven dairy farms in the UK whose pastures had received sewage sludge; PCBs were only positively identified in one sample at a concentration of 0.002 mg/kg.

LEVELS OF PCBs IN HUMAN FAT

35. In 1976–77 PCB levels were determined in 236 samples of human adipose tissue. Samples were obtained by necropsy from subjects over five years old during the course of routine examination by pathologists. The results are summarised below:

No. of Samples	*Sex*	*PCB Concentration (mg/kg)*	
		Arithmetic mean	*Range*
155	male	0.8	0.1 to 10
81	female	0.6	0.1 to 1.5

36. Prior to this study, measurements of PCBs had been made on only a few samples and levels had not exceeded 1 mg/kg. In this study, although the mean value is below 1 mg/kg, estimates exceeded this figure in about 16 per cent of the samples. Apart from a single high concentration of 10 mg/kg PCBs, found in the adipose tissue of a paint sprayer, the next highest sample was found to contain a concentration of 1.6 mg/kg. Similar increases have been observed in Norway and Finland. However, comparison with other studies show that PCB levels in human fat in the UK are among the lowest in Europe[20].

LEVELS OF PCBs IN HUMAN MILK

37. One hundred and two samples of whole human breast milk were analysed for contamination by PCBs in 1979 and 1980. Residues ranged from < 0.001–0.11 mg/kg in the whole milk, with a mean of 0.018 mg/kg, and the residues in milk fat were calculated to range from < 0.1–2.1 mg/kg with a mean of 0.5 mg/kg[21]. Levels of PCBs in human milk in the UK would therefore appear to be lower than those which have been obtained in studies undertaken in other countries[22,23] and in particular in the USA where a recent nationwide survey estimated the mean concentration of PCBs in breast milk fat to be 1.1 mg/kg[24].

38. The average likely daily intake of PCBs of breast-fed babies has been estimated to be approximately 15μg assuming a daily milk consumption of 810 ml per day[25].

CONCLUSIONS

39. On the basis of dietary surveys and by calculation it is estimated that human exposure in the UK to PCBs through the consumption of food is less than 40 micrograms per day and probably below 10 micrograms per day. The only food group consistently found to contain PCBs is the fish group and in people consuming greater than average amounts of fish (>500g/week), PCB consumption still did not exceed 40 micrograms per day. Infants receiving one teaspoon of fish liver oil daily are estimated to consume between 1 and 7 micrograms of PCBs daily from this source. The average likely daily intake of PCBs in breast-fed infants is estimated to be 15 micrograms.

40. In line with the results of surveys of PCBs in food in other countries, it has been found that in the UK PCB residues are almost ubiquitous to fish. Fish appear to be particularly susceptible to contamination because of the ability of the marine environment to act as a sink for discharges of PCBs. Conversely there is no evidence that PCB levels in freshwater fish caught in the UK approach anything like the very high levels which have been observed in some countries. This is almost certainly due in part to the lack of any large land-locked body of water in the UK which receives industrial effluents. The very low levels generally observed in marine species illustrate the capacity of UK coastal waters to disperse and effectively eliminate the effects of point source discharges of pollutants. Generally PCB concentrations in fish increased with increasing tissue lipid content. As far as other lipid-rich foodstuffs are concerned, the survey has shown that these are much less frequently found to contain residues of PCBs.

41. In view of the fact that PCBs may still be used in capacitors and transformers in food and feed manufacture, and in any case land-fill sites have received, and will continue to receive, articles containing PCBs, some leakage, spillage or other form of low-level discharge to the environment could still occur. Therefore further monitoring work will continue to be necessary from time to time.

REFERENCES

1. Chen, P H, Chang, K T and Lu, Y D (1981). Polychlorinated Biphenyls and Polychlorinated Dibenzofurans in the Toxic Rice-Bran Oil that caused PCB poisoning in Taichung. *Bull. Environ. Contam. Toxicol.,* **26,** 489–495.

2. World Health Organisation (1976). Polychlorinated Biphenyls and Terphenyls. *Environmental Health Criteria,* **2.**

3. Organisation for Economic and Cultural Development (1973). Polychlorinated biphenyls, their use and control. *Environmental Directorate, Paris.*

4. Zitko, V, Hutzingier, O and Safe, S (1971). Retention times and electron-capture detector responses of some individual chlorobiphenyls. *Bull. Environ. Contam. Toxicol.,* **6,** 160–163.

5. International Council for Exploration of the Seas (ICES) (1982). PCBs in the Marine Environment-An Overview. Report of the Advisory Committee on Marine Pollution, 1981. *ICES Co-operative Research Report* **112,** Annex 4, 43–50.

6. Department of the Environment (DOE) (1976). Polychlorinated Biphenyl (PCB) Wastes. *Waste Management Paper No. 6,* 19 pp.

7. Barber, C and Redhead, D L (1977). Maendy Landfill: A study of possible water pollution by leachate. *DOE WLR Technical Note Series,* **11,** 29 pp.

8. Barber, C, Young, C P, Naylor, J A and Connor, K J (1978). Brofiskin Landfill: A study of possible water pollution by leachate. *DOE WLR Technical Note Series,* **56,** 35 pp.

9. Nisbet, I C T and Sarofim, A F (1972). Rates and routes of transport of PCBs in the environment. *Env. Hlth. Persp.,* **1**(1), 21–38.

10. Portmann, J E (1979). Chemical monitoring of residue levels in fish and shellfish landed in England and Wales during 1970–73. *Aquat. Environ. Monit. Rep., MAFF Direct. Fish. Res. Lowestoft,* **1,** 70 pp.

11. Murray, A J (1979). Metals, organochlorine pesticides and PCB residue levels in fish and shellfish landed in England and Wales during 1974. *Aquat. Environ. Monit. Rep., MAFF Direct. Fish. Res. Lowestoft,* **2,** 11 pp.

12. ICES (1977). The ICES Co-ordinated Monitoring Programmes, 1975 and 1976. *ICES Co-operative Research Report,* **72,** 22 pp.

13. Ernst, W, Coerke, H, Eder, G and Shaefer, R G (1976). Residues of chlorinated hydrocarbons in marine organisms in relation to size and ecological parameters. I. PCB, DDT, DDE and DDD in fishes and molluscs from the English Channel. *Bull. Environ. Contam. Toxicol.,* **15**(1), 55–65.

14. ICES (1980). The ICES Co-ordinated Monitoring Programme, 1977. *ICES Co-operative Research Report*, **98**, 27 pp.

15. ICES (1983). The ICES Co-ordinated Monitoring Programmes, 1978 and 1979. *ICES Co-operative Research Report* (in press).

16. Kroner, R C (1971). *PCB Newsletter No 3,* 7, July 1971.

17. Lister, R E and Bennett, N J M (1972). PCBs in copying paper. *Nature,* **237,** 414.

18. Jelinek, C F, Braude, G L and Read, R B (1976). Paper presented at the Association of Metropolitan Sewerage Agencies Conference on Sludge Management, Houston, Texas. April 1976.

19. Environmental Canada Task Force Report, 1976.

20. Abbott, D C, Collins, G B, Goulding, R and Hoodless, R A (1981). Organochlorine pesticide residues in human fat in the United Kingdom, 1976–77. *Brit. Med. J.,* **283** (6304), 1425–1428.

21. Collins, G B, Holmes, D C and Hoodless, R A (1982). Organochlorine pesticide residues in human milk in Great Britain, 1979–80. *Human Toxicol.,* **1,** 425–431.

22. Polishuk, Z W, Ron, M, Wassermann, M, Cucos, S, Wassermann, D and Lemesch, C (1977). Pesticides in People. Organochlorine compounds in human blood plasma and milk. *Pesticides Monitoring Journal,* **10** (4), 121–129.

23. Atkinson, S A (1979). Chemical contamination of human milk: a review of current knowledge. *J. Canad. Diet. Assoc.,* **40**(3), 223–226.

24. Kennedy, D (1979). Polychlorinated biphenyls (PCBs); reduction of tolerances. *Federal Register,* **44** (127), 38330–38340.

25. Waterlow, J C and Thomson, A M (1979). Observations on the adequacy of breast feeding. *The Lancet,* II (8136), 238–242.

REPORT OF THE COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

1. We have been asked to consider the Report of the Steering Group on Food Surveillance — PCB Residues in Food and Human Tissues, and to advise the Food Additives and Contaminants Committee (FACC) on possible hazards to health from the levels of polychlorinated biphenyls (PCBs) found in food in the United Kingdom.

2. The Steering Group using data relevant to 1970–71, 1974 and 1976 has estimated by means of total diet studies the dietary intake of PCBs by the general population in the United Kingdom. Additional data presented relates to PCBs in a variety of fish and shellfish, fish liver oils, animal fats, milk, human milk[1] and human adipose tissue[2]. Samples of materials which come into contact with food have also been tested for possible PCB content. The food survey data clearly identify fish and shellfish as the major dietary sources of PCBs (many of the other food samples analysed contained no detectable amounts of PCBs i.e., < 0.01 mg/kg, and for milk < 0.002 mg/kg).

3. It is estimated that in the UK the average daily intake of PCBs from food is not greater than 40 μg and probably below 10 μg. In people consuming greater than average amounts of fish (> 500g/week) the PCB consumption was found to be not greater than 260 μg per week. Amounts of PCBs from fish liver oil supplements given to infants are estimated to provide between 1 μg and 7 μg daily.

COMMENTS

4. (a) Exposure to PCBs by the general population is almost entirely via dietary intake. Pre- and post-natal exposure of infants can occur due to PCBs crossing the placental barrier and also being excreted in milk[3].

(b) PCBs have produced acute and chronic toxicity in animal studies and man[4-9]. Data concerning toxic effects in man relate to episodes of accidental contamination of food (rice oil)[6-11] and industrial exposure[6]. Early clinical manifestations[7] are ocular discharge, oedema of eyelids, pigmentation of nails, mucous membranes and skin. General symptoms (abdominal, respiratory and neuro-muscular) tend to become more prominent later. Studies of the effects of PCBs in man and animals indicate that man appears to be the most sensitive species. The monkey is the only experimental species in which the qualitative and quantitative effects approach those observed in man[12]. The immune system of both animals and man has been shown to be affected by PCBs[12-15]. Fetal toxicity has also been observed in both animals and man[16-18]. Teratogenic effects have not been observed in man or the monkey following exposure to PCBs[16,17] but effects have been observed in the pig, dog and mouse[19-21]. Non-metastasising hepatocellular carcinomas have been demonstrated in mice and rats[22,23]. Evidence in man, based on epidemiological studies, is inadequate for assessing a carcinogenic effect[7,24].

PCBs accumulate in body tissues, especially adipose tissue, and the biological half-life in chronically exposed workers appears to be several months to 2–3 years[25]. The compounds with the highest degree of chlorination are retained the longest[26]. The monkey excretes PCBs at a rate very much slower than that found in the rat and it would appear that direct hydroxylation is not the major metabolic pathway in the monkey as is the case in the rat[27]. Hydroxylation is postulated to occur through an arene oxide intermediate[28]. It has been suggested that this differential rate and pathway of metabolism is the explanation of the species variation in response to the PCBs[27]. It is possible that different toxicological effects may result from differences in the degree of chlorination of the biphenyls[29].

(c) The exposure of adults and young infants to PCBs through food or breast milk is substantially below the minimum daily intake assessed as being toxic in the rice oil poisoning incident (2.8 mg/day for 135 days in a 50 kg adult)[30] and the dosage levels of Aroclor 1248, 2.5 to 5.0 mg/kg diet (approx. 0.1 to 0.2 mg/kg bodyweight/day) which produced overt toxic effects in rhesus monkey studies[31,32]. Studies carried out in primates do not permit the assessment of a no-effect level for PCBs. Furthermore the different toxic potentials of individual components of PCBs and the presence of impurities pose problems for establishing such a level.

(d) Commercial PCBs have been found to be contaminated both by polychlorinated dibenzofurans[33] and polychlorinated quaterphenyls[30,34]. Little is known about the toxicology of these compounds and they are likely to have been responsible, in part, for the effects seen in man. Substantially larger amounts of these impurities were present in the toxic rice oil causing human illness, than have been found in commercial PCBs or which are likely to be present in food or human tissues[35].

CONCLUSIONS

5. (a) We consider that the PCB content of the average national diet is unlikely to constitute a health hazard. Nevertheless animal studies suggest possible carcinogenic and teratogenic risks, that have not been ruled out as possible risks for humans. Such problems can only be resolved by prospective epidemiological studies.

(b) We concur with the conclusions of the Report and support the proposal for periodic monitoring in land-fill areas where human exposure to PCBs might be unacceptably high as a result of leaching from articles with PCB content causing contamination of the food chain.

(c) Furthermore as fish are particularly susceptible to PCB contamination we consider it advisable to monitor periodically samples of fish marketed in the United Kingdom.

(d) Information should be obtained on the intake of PCBs by pregnant and lactating women who consume large amounts of fish; and the resulting levels of PCBs in the milk of such mothers should be monitored as part of a further survey to determine the range of levels of PCBs in human milk in the United Kingdom.

(e) Every effort should be made to avoid accidental contamination of the food chain arising from the use of PCBs in capacitors and transformers especially in factories producing food or feeding stuffs.

REFERENCES

1. Collins, G B, Holmes, D C and Hoodless, R A (1982). Organochlorine pesticide residues in human milk in Great Britain, 1979–80. *Human Toxicol.*, **1**, 425–431.

2. Abbott, D C, Collins, G B, Goulding, R and Hoodless, R A (1981). Organochlorine pesticide residues in human fat in the United Kingdom, 1976–77. *Brit. Med. J.*, **283** (6304), 1425–1428.

3. Allen, J R and Barsotti, D A (1976). The effects of transplacental and mammary movement of PCBs on infant rhesus monkeys. *Toxicology*, **6**, 331–340.

4. Kimbrough, R, *et al* (1978). Animal Toxicology. *Environ. Hlth. Persp.*, **24**, 173–184.

5. IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans: Polychlorinated Biphenyls and Polybrominated Biphenyls, (1978) **18**, 70.

6. IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans: Polychlorinated Biphenyls and Polybrominated Biphenyls, (1978) **18**, 80–81.

7. National Conference on Polychlorinated Biphenyls. *Environmental Protection Agency; Office of Toxic Substances. Washington DC 20460.*, (1976), 14–29.

8. Chen, P H, Gaw, J M, Wong, G K and Chen, C J (1980). Levels and gas chromatographic patterns of polychlorinated biphenyls in the blood of patients after PCB poisoning in Taiwan. *Bull. Environ. Contam. Toxicol.*, **25**, 325–329.

9. Chen, P H, Chang, K T and Lu, Y D (1981). Polychlorinated biphenyls and polychlorinated dibenzofurans in the toxic rice-bran oil that caused PCB poisoning in Taichung. *Bull. Environ. Contam. Toxicol.*, **26**, 489–495.

10. Chang, K J *et al* (1980). Studies on patients with PCB poisoning. *J. Formosan Med. Assoc.*, **79**, 304–313.

11. Chang, K J *et al* (1980). Studies on patients with PCB poisoning. 2. Determination of urinary coproporphyrin, uroporphyrin, δ-aminolaevulinic acid and porphobilinogen. *Res. Comm. Chem. Pathol. Pharmacol.*, **30**, 547–553.

12. World Health Organisation (1976). Polychlorinated Biphenyls and Terphenyls. *Environmental Health Criteria*, **2**, 65–67.

13. Thomas, P and Hinsdill, R D (1978). Effect of polychlorinated biphenyls on the immune responses of rhesus monkeys and mice. *Toxicol. Appl. Pharmacol.*, **44**, 41–51.

14. Loose, L D *et al* (1977). Polychlorinated biphenyl and hexachlorobenzene induced humoral immunosuppression *J. Reticuloendothelial Soc.*, **22**(3), 253–271.

15. Silksworth, J B and Loose, L D (1979). PCB and HCB induced alterations of lymphocyte blastogenesis. *18th Annual Meeting of the Society of Toxicology. New Orleans. USA.*

16. IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans: Polychlorinated Biphenyls and Polybrominated Biphenyls, (1978) **18,** 71–72.

17. IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans: Polychlorinated Biphenyls and Polybrominated Biphenyls, (1978) **18**, 82.

18. Nakayama, K and Aoki, T (1977). Hazards of organic chlorine compounds to the health of children. *Paediatrician* **6**, 9–19.

19. Earl, F L, Couvillion, J L and Van Loon, E J (1974). The reproductive effect of PCB 1254 in beagle dogs and miniature-swine. *13th Annual Meeting of the Society of Toxicology. Washington DC. USA.,* (74).

20. Watanabe, M and Sugahara, T (1981). Experimental formation of cleft palate in mice with polychlorinated biphenyls (PCB). *Toxicology,* **19**(1), 49–53.

21. Lucier, G W, Davis, G J and McLachlan, J A, (1978). Transplacental toxicology of the polychlorinated polybrominated biphenyls. Developmental Toxicology of Energy Related Pollutants. *DOE symp. Ser.,* **47** (USA), 188–203.

22. Ito, N *et al* (1973). Histopathologic studies on liver tumorigenesis induced in mice by technical polychlorinated biphenyls and its promoting effect on liver tumours induced by benzene hexachloride. *J. Nat. Cancer Inst.,* **51**(5), 1637–1647.

23. Kimbrough, R D *et al* (1975). Induction of liver tumours in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. *J. Nat. Cancer Inst.,* **55** (6), 1453–1459.

24. IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans: Polychlorinated Biphenyls and Polybrominated Biphenyls, (1978) **18,** 82–83.

25. World Health Organisation (1976). Polychlorinated Biphenyls and Terphenyls. *Environmental Health Criteria,* **2**, 40.

26. Calabrase, E J and Sorensen, A J (1977). Review on Environmental Health. The Health Effects of PCBs., **2**(4), 287–288.

27. Allen, J E (1975). Response of the non-human primate to polychlorinated biphenyl exposure. *Federation Proc.,* **4**(8), 1675–1679.

28. Hsu, I C, Van Miller, J P, Seymour, J L and Allen, J R (1975). Urinary metabolites of 2,5,2′,5′ – tetrachlorobiphenyl in the non-human primate. *Proc. Soc. Exp. Biol. Med.,* **150**, 185–188.

29. Iatropoulos, M J *et al* (1978). Morphological Effects of 2,5,4′-Trichlorobiphenyl and 2,4,6,2′,4′-Pentachlorobiphenyl in Rhesus Monkeys. *Toxicol. Appl. Pharm.,* **45**(1), 338.

30. Hayabuchi, H, Yoshimura, T and Kuratsune, M (1979). Consumption of toxic rice oil by 'Yusho' patients and its relation to the clinical response and latent period. *Fd. Cosmet. Toxicol.,* **17**, 455–461.

31. National Conference on Polychlorinated Biphenyls (1976). *Environmental Protection Agency; Office of Toxic Substances. Washington DC 20460,* 43–49.

32. National Conference on Polychlorinated Biphenyls (1976). *Environmental Protection Agency; Office of Toxic Substances. Washington DC. 20460,* 459.

33. IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans: Polychlorinated Biphenyls and Polybrominated Biphenyls, (1978) **18**, 52–53.

34. Vos, J G *et al* (1970). Identification and toxicological evaluation of chlorinated dibenzofuran and chlorinated naphthalene in two commercial polychlorinated biphenyls. *Fd. Cosmet. Toxicol.,* **8**, 625–633.

35. Hori, S *et al* (1982). Effect of polychorinated biphenyls and polychlorinated quaterphenyls in Cynomologus monkey (*Macaca fascicularis*). *Toxicology,* **2** (24), 123–139.

CONSIDERATION OF THE REPORT BY THE FOOD ADDITIVES AND CONTAMINANTS COMMITTEE

The Food Additives and Contaminants Committee has considered a report by the Steering Group on Food Surveillance on PCB residues in food and human tissues and has made the following comments:

'The Committee notes that the exposure of the majority of the UK population to PCBs through the diet is low and recommends that the introduction of statutory controls on the maximum allowable levels of PCBs in food is not necessary at the present time.

In view of the fact that the principal source of PCBs in the diet is fish, the Committee endorses the conclusions of the Committee on Toxicity and recommends that samples of fish which are marketed in the UK continue to be monitored periodically.

The Committee notes that the Report of the Steering Group highlights a few localised instances where human exposure to PCBs might be unacceptably high such as in the vicinity of sites of disposal of PCBs. The Committee therefore recommends that monitoring should continue in land-fill areas where leaching from articles containing PCBs could result in the contamination of food.

The Committee recognises that human exposure might result from the accidental contamination of foods and feeding stuffs with PCBs as a result of their use in electrical equipment such as capacitors and transformers in the factory. The Committee recommends that these industries should take all possible steps to ensure that the likelihood of PCB contamination occurring via this route is minimised. The Committee has taken note of the action already being taken by the Health and Safety Executive in advising users of such electrical equipment of the risks and appropriate safety precautions to be taken'.

Printed in the UK for HMSO
Dd 736418 C15 8/83